WHAT IS CLAIMED IS:

1. A method for counteracting a pathologic change in a signal-transduction pathway involving a member of the steroid/thyroid hormone super-family, comprising administering to a mammalian subject in need an effective amount of a compound capable of inhibiting TGF-β signaling through a TGF-β receptor.

2. The method of claim 1 wherein the receptor is a steroid hormone receptor.

3. The method of claim 2 wherein the pathologic change is down- or up-regulation of the steroid hormone receptor.

4. The method of claim 3 wherein the down- or up-regulation involves TGF-β.

5. The method of claim 3 wherein the down- or up-regulation is induced by TGF-β.

6. The method of claim 1 wherein the pathologic change is a TGF-β induced change in the activity or signaling of a steroid hormone receptor.

7. The method of claim 2 wherein the steroid hormone receptor is glucocorticoid receptor.

8. The method of claim 1 wherein the receptor is a thyroid hormone receptor.

9. The method of claim 8 wherein the pathologic change is down- or up-regulation of a thyroid hormone receptor.

10. The method of claim 9 wherein the down- or up-regulation involves TGF-β.

11. The method of claim 9 wherein the down- or up-regulation is induced by TGF-β.

12. The method of claim 8 wherein the pathologic change is a TGF-β induced change in the activity or signaling of a thyroid hormone receptor.

13. The method of claim 1 wherein the receptor is a retinoic acid receptor.

14. The method of claim 13 wherein the pathologic change is down- or up-regulation of a retinoic acid receptor.

15. The method of claim 14 wherein the down- or up-regulation involves TGF-β.

16. The method of claim 14 wherein the down- or up-regulation is induced by TGF-β.

17. The method of claim 13 wherein the pathologic change is a TGF-β induced change in the activity or signaling of a retinoic acid receptor.

18. The method of claim 1 wherein the TGF-β receptor is a TGFβ-R1 kinase.

19. The method of claim 18 wherein the compound is capable of binding to said TGFβ-R1 kinase.

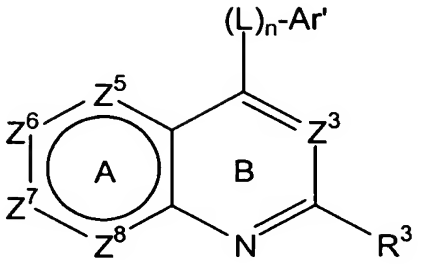
20. The method of claim 19 wherein the compound is capable of binding to an additional receptor kinase.

21. The method of claim 20 wherein the additional receptor kinase is an activin receptor (Alk4).

22. The method of claim 1 wherein the compound is a non-peptide small molecule.

23. The method of claim 22 wherein the compound is a small organic molecule.

24. The method of claim 23 wherein the small organic molecule is a compound of formula (1)



(1)

or the pharmaceutically acceptable salts thereof

wherein R^3 is a noninterfering substituent;

each Z is CR^2 or N, wherein no more than two Z positions in ring A are N, and wherein two adjacent Z positions in ring A cannot be N;

each R^2 is independently a noninterfering substituent;

L is a linker;

n is 0 or 1; and

Ar' is the residue of a cyclic aliphatic, cyclic heteroaliphatic, aromatic or heteroaromatic moiety optionally substituted with 1-3 noninterfering substituents.

25. The method of claim 24 wherein the compound is a quinazoline derivative.

26. The method of claim 25 wherein Z^3 is N; and Z^5-Z^8 are CR^2.

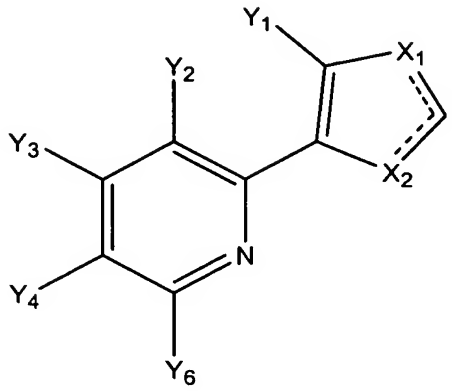
27. The method of claim 25 wherein Z^3 is N; and at least one of Z^5-Z^8 is nitrogen.

28. The method of claim 25 wherein R^3 is an optionally substituted phenyl moiety.

29. The method of claim 28 wherein R^3 is selected from the group consisting of 2-, 4-, 5-, 2,4- and 2,5-substituted phenyl moieties.

30. The method of claim 29 wherein at least one substituent of the phenyl moiety is an alkyl(1-6C), or halo.

31. The method of claim 23, wherein the small organic molecule is a compound of formula (2)

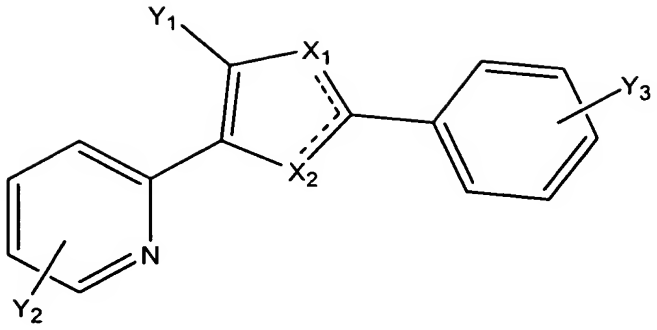


wherein Y_1 is phenyl or naphthyl optionally substituted with one or more substituents selected from halo, alkoxy(1-6 C), alkylthio(1-6 C), alkyl(1-6 C), haloalkyl (1-6C), -O-$(CH_2)_m$-Ph, -S-$(CH_2)_m$-Ph, cyano, phenyl, and CO_2R, wherein R is hydrogen or alkyl(1-6 C), and m is 0-3; or phenyl fused with a 5- or 7-membered aromatic or non-aromatic ring wherein said ring contains up to three heteroatoms, independently selected from N, O, and

Y_2, Y_3, Y_4, and Y_5 independently represent hydrogen, alkyl(1-6C), alkoxy(1-6 C), haloalkyl(1-6 C), halo, NH_2, NH-alkyl(1-6C), or $NH(CH_2)_n$-Ph wherein n is 0-3; or an adjacent pair of Y_2, Y_3, Y_4, and Y_5 form a fused 6-membered aromatic ring optionally containing up to 2 nitrogen atoms, said ring being optionally substituted by one or more substituents independently selected from alkyl(1-6 C), alkoxy(a-6 C), haloalkyl(1-6 C), halo, NH_2, NH-alkyl(1-6 C), or $NH(CH_2)_n$-Ph, wherein n is 0-3, and the remainder of Y_2, Y_3, Y_4, and Y_5 represent hydrogen, alkyl(1-6 C), alkoxy(1-6C), haloalkyl(1-6 C), halo, NH_2, NH-alkyl(1-6 C), or $NH(CH_2)_n$-Ph wherein n is 0-3; and

one of X_1 and X_2 is N and the other is NR_6, wherein R_6 is hydrogen or alkyl(1-6 C).

32. The method of claim 23 wherein the small organic molecule is a compound of formula (3)



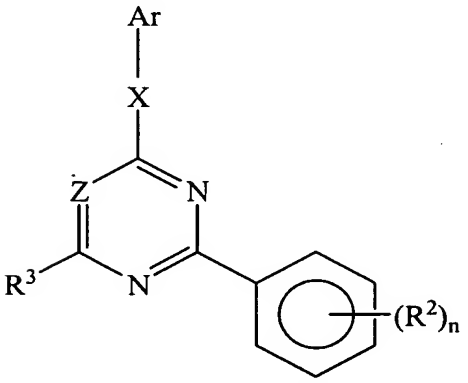
wherein Y_1 is naphthyl, anthracenyl, or phenyl optionally substituted with one or more substituents selected from the group consisting of halo, alkoxy(1-6 C), alkylthio(1-6 C), alkyl(1-6 C), -O-(CH_2)-Ph, -S-$(CH_2)_n$-Ph, cyano, phenyl, and CO_2R, wherein R is hydrogen or alkyl(1-6 C), and n is 0, 1, 2, or 3; or Y_1 represents phenyl fused with an aromatic or non-aromatic cyclic ring of 5-7 members wherein said cyclic ring optionally contains up to two heteroatoms, independently selected from N, O, and S;

Y_2 is H, $NH(CH_2)_n$-Ph or NH-alkyl(1-6 C), wherein n is 0, 1, 2, or 3;

Y_3 is CO_2H, $CONH_2$, CN, NO_2, alkylthio(1-6 C), -SO_2-alkyl(C1-6), alkoxy(C1-6), $SONH_2$, CONHOH, NH_2, CHO, CH_2NH_2, or CO_2R, wherein R is hydrogen or alkyl(1-6 C);

one of X_1 and X_2 is N or CR', and other is NR' or CHR' wherein R' is hydrogen, OH, alkyl(C-16), or cycloalkyl(C3-7); or when one of X_1 and X_2 is N or CR' then the other may be S or O.

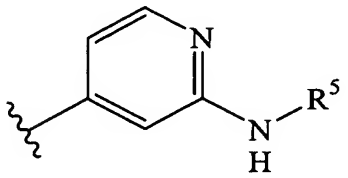
33. The method of claim 23 wherein the small organic molecule is a compound of formula (4)



(4)

and the pharmaceutically acceptable salts and prodrug forms thereof; wherein

Ar represents an optionally substituted aromatic or optionally substituted heteroaromatic moiety containing 5-12 ring members wherein said heteroaromatic moiety contains one or more O, S, and/or N with a proviso that the optionally substituted Ar is not



wherein R^5 is H, alkyl (1-6C), alkenyl (2-6C), alkynyl (2-6C), an aromatic or heteroaromatic moiety containing 5-11 ring members;

X is NR^1, O, or S;

R^1 is H, alkyl (1-8C), alkenyl (2-8C), or alkynyl (2-8C);

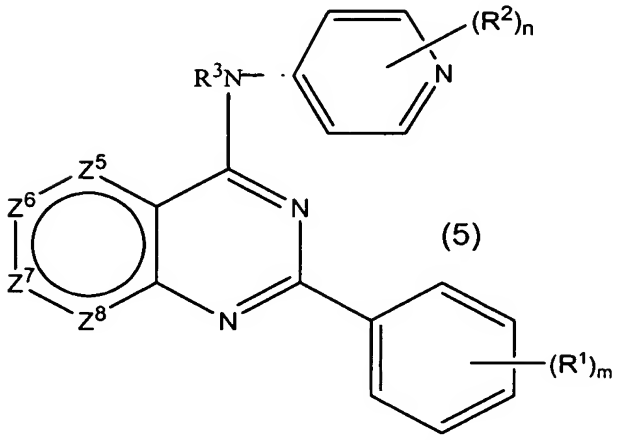
Z represents N or CR^4;

each of R^3 and R^4 is independently H, or a non-interfering substituent;

each R^2 is independently a non-interfering substituent; and

n is 0, 1, 2, 3, 4, or 5. In one embodiment, if n>2, and the R^2's are adjacent, they can be joined together to form a 5 to 7 membered non-aromatic, heteroaromatic, or aromatic ring containing 1 to 3 heteroatoms where each heteroatom can independently be O, N, or S.

34. The method of claim 23 wherein the small organic molecule is a compound of formula (5)



(5)

or the pharmaceutically acceptable salts thereof;

wherein each of Z^5, Z^6, Z^7 and Z^8 is N or CH and wherein one or two Z^5, Z^6, Z^7 and Z^8 are N and wherein two adjacent Z positions cannot be N;

wherein m and n are each independently 0-3;

wherein two adjacent R^1 groups may be joined to form an aliphatic heterocyclic ring of 5-6 members;

wherein R^2 is a noninterfering substituent; and

wherein R^3 is H or CH_3.